

Photo Quiz: Sudden Death

Isaac H. Solomon, a,b Robert F. Padera, Jr.a,b

^aDepartment of Pathology, Brigham and Women's Hospital, Boston, Massachusetts, USA

KEYWORDS pulmonary embolism

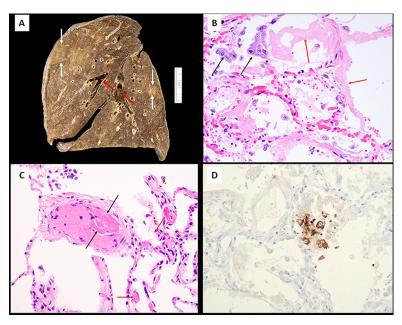


FIG 1 (A) Gross photograph of a parasagittal section of the right lung, demonstrating large central pulmonary emboli (red arrows) and patchy areas of consolidation (white arrows). (B) High-power photomicrograph of a hematoxylin and eosin-stained section of lung demonstrating hyaline membranes (red arrows) and reactive pneumocytes (black arrows). (C) High-power photomicrograph of a hematoxylin and eosin-stained section of lung demonstrating microthrombi within capillaries (red arrows) and venules (black arrows). (D) High-power photomicrograph of diagnostic viral immunohistochemical stain of lung. The original magnification for images B, C, and D was $\times 400$.

he patient was a 70-year-old African-American man from Boston, MA, who presented in April 2020 with a 10-day history of cough and progressive shortness of breath, with fevers over the last 3 days. He had had no recent travel but resided with two family members who had exhibited similar symptoms. His past medical history was significant for poorly controlled hypertension, obesity, and type 2 diabetes. He called 911 for acute worsening of severe shortness of breath. Upon arrival of EMS, his vital signs were a heart rate of 121 beats per minute, a respiratory rate of 32 breaths per minute, a temperature of 38.3°C, blood pressure of 146/90 mmHg, and oxygen saturation of 81%. He was brought to the hospital emergency department, where a chest X-ray showed bilateral interstitial opacities, and a bedside echocardiogram revealed right heart strain. He developed a pulseless electrical activity arrest 1 h after arrival at the hospital and was unable to be resuscitated. Laboratory work revealed a D-dimer level of >5,000 ng/ml (reference range, <500 ng/ml), a ferritin level of 724 μ g/ liter (reference range, 30 to 400 µg/liter), a lactate dehydrogenase level of 357 U/liter

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Address correspondence to Robert F. Padera, Jr., rpadera@bwh.harvard.edu.

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^bDepartment of Pathology, Harvard School of Medicine, Boston, Massachusetts, USA

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(reference range, 135 to 225 U/liter), a white blood cell count of $12,600/\mu$ l (reference range, 4,000 to $10,000/\mu$ l) with 11% lymphocytes (reference range, 18 to 41%), a platelet count of $114,000/\mu$ l (reference range, 150,000 to 450,000/ μ l), and negative blood cultures.

A complete autopsy with full anatomic dissection was performed, in which organs including the brain were removed, dissected, and examined. Tissue samples for histology were fixed in 10% formalin overnight prior to processing. Gross examination revealed large bilateral pulmonary thromboemboli (Fig. 1A) and additional thromboemboli coiled within the right atrium. Histologic evaluation of the lungs revealed the exudative phase of diffuse alveolar damage, with hyaline membranes, reactive pneumocyte hyperplasia with occasional multinucleated forms, increased intra-alveolar macrophages, and mild interstitial chronic inflammation (Fig. 1B), corresponding to the areas of gross consolidation. No clear viral cytopathic effects were identified, and no evidence of bronchopneumonia was present. In addition to the large pulmonary thromboemboli seen grossly, there was microscopic evidence of microvascular thrombosis in alveolar capillaries and venules (Fig. 1C). A specimen from the postmortem examination was sent for diagnostic testing, and immunohistochemistry for viral organisms was performed (Fig. 1D).